Table CDC status and peripheral blood CD4+ and CD8+ lymphocyte counts in HIV-infected homosexual/bisexual subjects presenting with oral hairy leukoplakia (OHL) and progressing to AIDS by eighteen months

Presenting with OHL				*Progressing to AIDS*		
		Mean & median T-cell counts/ul (range)*			*Mean & median* T-cell counts/ul (range)*	
CDC status	*Number (%)*	*CD4+*	*CD8+*	*Number (%)*	*CD4+*	*CD8+*
II	2 (6)	240	760	1 (8)	200	510
III	13 (37)	670	860	4 (34)	190	730
IV-A, C2	20 (57)	450	550	7 (58)	450	550
		490	650		260	660
Totals	35 (100)	500* (150–1100)	650* (200–1300)	12 (100)	130* (50–620)	800* (200–1000)

*Median values of lymphocyte counts obtained from all the subjects in each column.

We interrogated the cohort database for all HIV-infected subjects from the onset of OHL, together with details of their subsequent clinical behaviour. Having excluded those with an initial diagnosis of AIDS (CDC IV-C1, B, D, E) and on anti-retroviral treatment, there were 35 cases of OHL in the remaining categories (table). Most of these were in IV-C2, but a substantial proportion (43%) would otherwise have been classified CDC II and III.

The peripheral CD4+ count of subjects presenting with OHL displayed a wide range of values, but as a group, the CD4+ count deteriorated markedly as AIDS developed. The progression rate for these 35 subjects with OHL was 33% at 12 months and 83% at 36 months, comparable to rates determined by Kelly *et al*[1] and Greenspan *et al*.[5] However, we identified four subjects with OHL (11%) who have been followed up for more than 36 months (mean 42·5, range 41–56 months), and who have not progressed to AIDS. One subject is in CDC II, the remainder in CDC III. These individuals may represent a sub-group of OHL-bearing subjects in which the severity of immunodeficiency is not as marked as in those with oral candida, for example, and in whom the rate of immunological decline seems slower. In classifying patients with OHL alone in CDC IV-C2, this heterogeneity, which may be reflected in their subsequent clinical behaviour, is lost.

In the light of current trends for earlier intervention with anti-retroviral and prophylactic agents, the clinical presence of OHL serves as an earlier (albeit "softer") marker of immunosuppression than oral candida. We suggest that OHL should not necessarily be regarded as an ominous sign, but instead should prompt additional clinical and laboratory assessment of possible HIV disease progression.

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Antiviral chemotherapy for retinitis in HIV-infected patients

Millar *et al*[1] are uncertain why zidovudine has apparently produced regression of retinitis in patients with the acquired immune deficiency syndrome (AIDS).[2-4] They note that one possible explanation is a bidirectional interaction between cytomegalovirus (CMV) and human immunodeficiency virus (HIV) leading to down regulation of CMV replication when zidovudine inhibits HIV replication.[5] Alternatively, HIV itself may play a more direct role in the development of retinitis than is currently supposed.[6]

Retinitis in patients with AIDS is attributed to CMV without proof that the association is causal.[6] HIV-induced damage to neuroretinal cells could be the initiating event of retinitis in these patients, with CMV playing either no or only a secondary role.[6] The clinical responses in AIDS-related retinitis produced in uncontrolled trials by the anti-CMV agent ganciclovir fail to establish either that the drug is effective[7] or that the retinitis is caused by CMV.[6] Anti-HIV chemotherapy alone or combined with anti-CMV chemotherapy could be more effective than anti-CMV chemotherapy alone in HIV-infected patients with retinitis. As assessment of combined systemic ganciclovir and zidovudine therapy is precluded by haematological toxicity,[1] the role of adjunctive anti-HIV chemotherapy in retinitis will only be defined when less toxic anti-CMV agents or intravitreal ganciclovir therapy are used.

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Gastrointestinal obstruction associated with *Chlamydia trachomatis*

I read with great interest the recent report of Pegg and Owen regarding small bowel obstruction associated with *Chlamydia trachomatis*.[1] I would like to point out, however, that this phenomenon was first suggested in a similar case report in 1987,[2] and recently included in a review of abdominal pain syndromes caused by chlamydial infections.[3]

While both cases[1,2] presented clinical, historical and serologic evidence of a chlamydial aetiology for the small bowel obstruction, the report of Pegg and Owen additionally demonstrated *C trachomatis* in the genital tract by ELISA testing.

The women in both reports had no genital tract complaints at the time of presentation, and their fallopian tubes appeared normal. Both of these cases strongly suggest that infection with *C trachomatis* may result in small bowel obstruction, and that pelvic symptoms may not be temporally associated with the abdominal disease.

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Value of performing biopsies in genitourinary clinics

We read with interest the recent letter in your journal concerning the value of performing biopsies in genitourinary clinics.[1]

Men with abnormalities of the skin of the penis frequently present to genitourinary physicians, who must therefore also be skilled in dermatology. This is particularly so in the light of the probable re-definition of our specialty as "dermato-venereology" in the unified post-1992 European Community.

In order to assess the specific dermatological problems encountered by a busy genitourinary medicine clinic, we set up an internal clinic for penile dermatoses. The criteria for referral were a penile dermatosis of uncertain diagnosis for which the clinician thought that a biopsy might prove helpful. The technique used was that of local anaesthesia followed by skin snip biopsy. All patients were thoroughly counselled before the procedure, which was generally regarded by the patient as not being as traumatic as initially envisaged.

Over a period of two months a total of 18 biopsies were performed with the following histopathological results: 5 nonspecific dermatitis, 3 viral warts, 3 lichen sclerosus et atrophicus, 2 lichen planus, 2 symptomatic subclinical papillomavirus infection, 1 granulomatous disease (currently undergoing investigation) and 1 trauma (probable dermatitis artefacta). Although only a small sample was taken, typically 0·5 mm in diameter, in only one case did the biopsy prove non-diagnostic.

It can be seen that in 11 out of 18 of cases, a specific diagnosis was able to be made for which a management plan could be devised. We would draw particular attention to the diagnoses of lichen sclerosus et atrophicus (LSA). Not only are there multiple names for the same histopathological condition (LSA, balanitis xerotica obliterans and kraurosis vulvae), but the malignant potential of this common[2] condition remains undefined and a standard text[3] suggests six to twelve monthly follow-up for life.

Despite the longstanding combination of the specialties of dermatology and venereology on the continent, the literature on genital dermatology is scant. The three specialties of genitourinary medicine, dermatology and urology have overlapping interests in penile cutaneous disorders, but rarely have in-depth knowledge. Unlike the vulval cutaneous disorders,[4] for example, there is no standard textbook in the English language on penile dermatoses. Furthermore, confusions still exist over relatively common disorders, as described above. It is thus important to develop and improve lines of communication between genitourinary physicians, dermatologists and histopathologists at regular audit meetings.

We would therefore wholeheartedly agree with the conclusion of Drs Arumainayagam and Sumathipala that penile biopsy is a very useful diagnostic procedure in the setting of a genitourinary clinic. The more widespread use of this simple and minimally invasive procedure would allow us to gain greater insight into the ill-understood incidence and nature of genital dermatoses.

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Sexual assault of men: a series

The sexual assault of men has attracted little attention. The report of Hillman and colleagues describes five cases of male sexual assault from two large genitourinary medicine (GUM) departments during an unspecified time period.[1] We suspect their report is not representative of men attending GUM departments after sexual assault and write to report our experience.

During 1989, 10 male patients attended this department reporting penetrative sexual assault by men. They presented four days to one year after the assault and patient details are